WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Monday, August 30, 2004

Hide?	Set Name	Query	Hit Count
		DB=USPT,EPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L1	shoshan-a$.in. or wasserman-A$.in. or Mintz-E$.in. or mintz-L$.in. or faigler-S$.in.	114
☐	L2	transcriptome	54
☐	L3	RNA transcript	6214
☐	L4	alternativ$ spliced gene$1	31
☐	L5	penn-S$.in. or Rank-D$.in. or Hanzel-d$.in.	151
☐	L6	libar$3 and L2	0
☐	L7	L1 and L2	1
☐	L8	L1 and L4	1
☐	L9	L5 and L4	0
☐	L10	L5 and L3	1
☐	L11	RNA and L5	1
☐	L12	penn-Sharron$.in.	5
☐	L13	transcriptome	54
☐	L14	RNA transcript	6214
☐	L15	Alternativ$ spliced (genes or transcript or sequence)	275
☐	L16	(L13 or L14 or L15) and microarray	634
☐	L17	L16 and human	622
☐	L18	L17 and (plurality near (oligonucleotide or probe))	20
☐	L19	L17 and (high-throughput)	193
☐	L20	L19 and library	189
☐	L21	L18 and (messenger RNA or mRNA)	19
☐	L22	(plurality same (probe or oligonucleotide))	19074
☐	L23	L22 and L20	19
☐	L24	L2 and (microarray or array)	26
☐	L25	alternativ$ splic$ near (genes or sequence or transcript)	574
☐	L26	pool near RNA transcript	3
☐	L27	(plurality or pool) near (messenger RNA or mRNA)	337
☐	L28	L27 same microarray	6
☐	L29	L25 same microarry	1

☐	L30	L25 same microarray	3
☐	L31	L25 and gene expression profile	12
☐	L32	L27 and gene expression profile	18
☐	L33	RNA transcripts and differential display	270
☐	L34	L33 and L25	26
☐	L35	L34 and library	26
☐	L36	shoshan-a$.in. or wasserman-A$.in. or Mintz-E$.in. or mintz-L$.in. or faigler-S$.in.	114
☐	L37	transcriptome	54
☐	L38	RNA transcript	6214
☐	L39	alternativ$ spliced gene$1	31
☐	L40	penn-S$.in. or Rank-D$.in. or Hanzel-d$.in.	151
☐	L41	libar$3 and L37	0
☐	L42	L36 and L37	1
☐	L43	L36 and L39	1
☐	L44	L40 and L39	0
☐	L45	L40 and L38	1
☐	L46	RNA and L40	1
☐	L47	penn-Sharron$.in.	5
☐	L48	transcriptome	54
☐	L49	RNA transcript	6214
☐	L50	Alternativ$ spliced (genes or transcript or sequence)	275
☐	L51	(L48 or L49 or L50) and microarray	634
☐	L52	L51 and human	622
☐	L53	L52 and (plurality near (oligonucleotide or probe))	20
☐	L54	L52 and (high-throughput)	193
☐	L55	L54 and library	189
☐	L56	L53 and (messenger RNA or mRNA)	19
☐	L57	(RNA transcript) and @pd > 20030424	1033
☐	L58	(plurality same (probe or oligonucleotide))	19074
☐	L59	L57 and L55	81
☐	L60	L37 and (microarray or array)	26
☐	L61	alternativ$ splic$ near (genes or sequence or transcript)	574
☐	L62	(L47 and (microarray or array)) and @pd > 20030424	1
☐	L63	(plurality or pool) near (messenger RNA or mRNA)	337
☐	L64	((plurality or pool) near (messenger RNA or mRNA)) and @pd > 20030424	57
☐	L65	L60 and gene expression profile	7
☐	L66	RNA transcripts and differential display	270

DB=PGPB,USPT,USOC,EPAB,DWPI; PLUR=YES; OP=ADJ

☐	L67	splic$ variant same unique sequence	20
☐	L68	L67 same (oligonucleotide or array or microarray or transciptome)	0
☐	L69	library same (set near splice variant)	1
☐	L70	library same (splice variants)	930
☐	L71	L70 same (unique sequence)	0
☐	L72	L70 same (mRNA or messenger RNA)	539
☐	L73	L72 and unique sequence	24
☐	L74	splice$ variant same unique sequence	20
☐	L75	(set or plurality or multiple) near (splic$ variant)	179
☐	L76	L75 same ((single or one) near (transcription unit or gene or transcriptome or sub-transcriptome))	8
☐	L77	L75 same (unique sequenc$)	0
☐	L78	L75 and(unique sequenc$)	13
☐	L79	L74 and (microarray or array)	15
☐	L80	L78 AND (MICROARRAY OR ARRAY)	7
☐	L81	L75 SAME (microarray)	0
☐	L82	L75 AND (microarray)	62
☐	L83	SET SAME (SPLIC$ VARIANT)	677
☐	L84	SET NEAR (SPLIC$ VARIANT)	47
☐	L85	ALTERNAT$ SPLIC$ NEAR UNIQU$ SEQUENC$	0
☐	L86	ALTERNATIVE SPLICED vARIANTS	13
☐	L87	SPLICED vARIANTS	668
☐	L88	L87 SAME ((TIL$ OR OVERLAP$) NEAR PROB$)	1
☐	L89	L83 SAME ((TIL$ OR OVERLAP$) NEAR PROB$)	1
☐	L90	(L83 or l87)SAME ((TIL$ OR OVERLAP$) NEAR (PROB$ or sequenc$))	15
☐	L91	(L83 or l87) and ((TIL$ OR OVERLAP$) NEAR (PROB$ or sequenc$))	442
☐	L92	L91 and unique sequenc$	27

END OF SEARCH HISTORY

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      (FILE 'HOME' ENTERED AT 16:10:53 ON 30 AUG 2004)

      FILE 'MEDLINE, BIOTECHDS, EMBASE, BIOSIS, SCISEARCH, CANCERLIT, CAPLUS'
      ENTERED AT 16:11:22 ON 30 AUG 2004
L1          3127 S ALTERNATIV? SPLIC? VARIAN?
L2            13 S L1 AND UNIQUE SEQUENC?
L3             0 S L1 AND (TIL? SEQUENC? OR OVERLAP? SEQUENC?)
L4          2102 S L1 AND (TRANSCRIPTOME OR TRANSCRIPTION UNIT OR EXON OR GENE)
L5          2102 S L4 (10A)  (TRANSCRIPTOME OR TRANSCRIPTION UNIT OR EXON OR GE
L6           655 S L1 (10A) (TRANSCRIPTOME OR TRANSCRIPTION UNIT OR EXON OR GENE
L7           454 S L1 (5A) (TRANSCRIPTOME OR TRANSCRIPTION UNIT OR EXON OR GENE)
L8           170 DUP REM L7 (284 DUPLICATES REMOVED)
L9             5 S L8 AND ((SET OR MULTIPLE OR PLURALITY) (3A) SPLIC### VARIAN#
L10          234 S L1 AND UNIQUE
L11           66 DUP REM L10 (168 DUPLICATES REMOVED)
L12            0 S L1 AND EXON PROFILING ARRAY
L13           15 S L1 AND MICROARRAY
L14           10 DUP REM L13 (5 DUPLICATES REMOVED)
L15         8945 S TRANSCIPTOME OR TRANSCRIPTION UNIT
L16            1 S L15 AND L1
L17         8945 S TRANSCIPTOME OR TRANSCRIPTION UNIT OR SUBTRANSCRIPTOME
L18            1 S L17 AND L1
L19          531 S OVERLAPPING SEQUENCE OR TILING SEQUENCE
L20            0 S L1 AND L19
L21        28419 S SINGLE GENE OR SINGLE GENOME
L22           49 S L21 AND L1
L23           14 DUP REM L22 (35 DUPLICATES REMOVED)

      FILE 'STNGUIDE' ENTERED AT 16:39:49 ON 30 AUG 2004
L24            0 S UNIQUE SEQUENCE
L25            0 S UNIQUE SEQUENC?
L26            0 S UNIQUE (3A) SEQUENC?
L27            0 S LIBRAR? AND L1

      FILE 'MEDLINE, BIOTECHDS, EMBASE, BIOSIS, SCISEARCH, CANCERLIT, CAPLUS'
      ENTERED AT 16:48:35 ON 30 AUG 2004
L28         8784 S UNIQUE SEQUENC?
L29           13 S L28 AND L1
L30       316787 S LIBRAR?
L31          248 S L30 AND L1
L32           88 DUP REM L31 (160 DUPLICATES REMOVED)
L33           31 S L31 AND UNIQUE
L34           10 DUP REM L33 (21 DUPLICATES REMOVED)

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Alternative splicing generates multiple isoforms of a rabbit prostaglandin E2 receptor.

AUTHOR: Breyer R M; Emeson R B; Tarng J L; Breyer M D; Davis L S; Abromson R M; Ferrenbach S M

CORPORATE SOURCE: Division of Nephrology, Vanderbilt University School of Medicine, Nashville, Tennessee.

CONTRACT NUMBER: DK-39261-05 (NIDDK)
DK-46205-01 (NIDDK)

SOURCE: Journal of biological chemistry, (1994 Feb 25) 269 (8) 6163-9.
Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-U04273; GENBANK-U04274; GENBANK-U04275; GENBANK-U04276

ENTRY MONTH: 199404

ENTRY DATE: Entered STN: 19940412
Last Updated on STN: 19940412
Entered Medline: 19940401

AB Four cDNA clones homologous with a murine prostaglandin E2 receptor have been isolated from a rabbit kidney cortex cDNA library. These cDNAs encode related proteins that differ only in their COOH-terminal sequences. Southern blot analysis of rabbit genomic DNA indicates that these receptor cDNAs represent **alternatively spliced variants** derived from a single gene. This was confirmed by isolation and sequence analysis of genomic clones containing common region exons and **unique** 3'-coding exons, which contained intron/exon boundaries at the predicted splice junctions. Transient expression of a novel full-length cDNA in COS1 cells confirmed the ligand binding profile typical of an EP3 receptor subtype. Ribonuclease protection assays indicate that the gene encoding these receptors is most highly expressed in kidney, adrenal, and stomach with lower but significant expression in uterus, lung, heart, ileum, spleen, and brain. Moreover, each of the cloned isoforms is expressed in the kidney. In situ hybridization analyses of rabbit kidney demonstrated that the level of expression is highest in the outer medulla with lesser expression in the cortex and no detectable expression in the inner medulla. The ligand binding profile and tissue distribution of these receptors is consistent with a functional role for this family of EP3 receptors in mediating the renal actions of prostaglandins as well as the effects of prostaglandins on gastric acid secretion and adrenal function.

L11 ANSWER 64 OF 66 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1993:664042 CAPLUS

DOCUMENT NUMBER: 119:264042

TITLE: Enhanced activation of the human histone H2B promoter by an Oct-1 variant generated by alternative splicing

AUTHOR(S): Das, Gokul; Herr, Winship

CORPORATE SOURCE: Cold Spring Harbor Lab., Cold Spring Harbor, NY, 11724, USA

SOURCE: Journal of Biological Chemistry (1993), 268(33), 25026-32
CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal

LANGUAGE: English

AB POU homeodomain proteins are important regulators of ubiquitous as well as tissue-sp. transcription. These factors include the broadly expressed octamer motif-binding protein Oct-1 and the related cell-sp. expressed protein Oct-2. These two proteins differ in the types of octamer motif-containing promoters they preferentially activate; Oct-1 can activate RNA polymerase II transcription from a small nuclear RNA promoter better than Oct-2, which can better activate an mRNA-type promoter. The authors

describe a variant Oct-1-encoding cDNA resulting from two sep. alternate splices of the human oct-1 primary transcript; these alternate splices were present in all cell lines tested. This cDNA encodes an amino-terminally and carboxyl-terminally truncated form of Oct-1, called Oct-1B, which retains the DNA-binding POU domain and acquires a **unique** 12-amino acid carboxyl-terminal extension. In a transient expression assay, Oct-1B displayed an enhanced ability compared to the larger form of Oct-1 (called Oct-1A in this report) to activate the human histone H2B promoter, an mRNA-type promoter where a natural octamer motif is involved in cell cycle dependent transcription. Thus, the ability of Oct-1 related proteins to activate a natural regulatory target can be influenced by alternative splicing.

L11 ANSWER 65 OF 66 MEDLINE on STN DUPLICATE 45
ACCESSION NUMBER: 93352614 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7688734
TITLE: Two truncated forms of rat insulin receptor-related receptor.
AUTHOR: Itoh N; Jobo K; Tsujimoto K; Ohta M; Kawasaki T
CORPORATE SOURCE: Department of Biological Chemistry, Kyoto University Faculty of Pharmaceutical Sciences, Japan.
SOURCE: Journal of biological chemistry, (1993 Aug 25) 268 (24) 17983-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D13965; GENBANK-D13966
ENTRY MONTH: 199309
ENTRY DATE: Entered STN: 19931001
Last Updated on STN: 20000303
Entered Medline: 19930916

AB The insulin receptor-related receptor (IRR) (1271 amino acids) is expected to have **unique** functions as a novel member of the insulin receptor family. In this paper, we report two **alternatively spliced variants** of rat IRR mRNA, which are predicted to encode two truncated forms of IRR, sIRR-1 (410 amino acids) and sIRR-2 (469 amino acids). The amino acid sequence of sIRR-1 is identical to the N-terminal 410-amino acid sequence of IRR. sIRR-2 has an additional 59-amino acid insertion in the C-terminal region. Both truncated forms retain the N-terminal and cysteine-rich domains but lack the transmembrane and intracellular tyrosine kinase domains, indicating that the truncated forms are the secreted forms. The translation products of the truncated form mRNAs were detected in the stomach and kidney by Western analysis. However, the physiological significance of the secreted forms remains to be elucidated.

Splice variants of a metabotropic glutamate receptor

INVENTOR(S): Savitzky, Kinneret; Toporik, Amir; Mintz, Liat
PATENT ASSIGNEE(S): Israel

SOURCE: U.S. Pat. Appl. Publ., 60 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002081655	A1	20020627	US 2001-871874	20010604
PRIORITY APPLN. INFO.:			IL 2000-136553 A	20000605

AB The present invention provides nucleic acid sequences encoding **alternative splicing variants** of a metabotropic glutamate receptor (mGluR). The invention also provides amino acid sequences coded by the isolated nucleic sequences purified antibodies which bind specifically to the splicing isoform, and expression vectors comprising any one of the nucleic acid sequences. The invention also provides pharmaceutical compns. comprising as an active ingredient the expression vector or an amino acid sequence.

expression in response to a rise in intracellular Ca2+.
AUTHOR: Zacharias D A; Strehler E E
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Mayo Graduate School, Mayo Clinic/Foundation, Rochester, Minnesota 55905, USA.
SOURCE: Current biology : CB, (1996 Dec 1) 6 (12) 1642-52. Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970306
Last Updated on STN: 19970306
Entered Medline: 19970227

AB BACKGROUND: Most eukaryotic genes are divided into introns and exons. Upon transcription, the intronic segments are eliminated and the exonic sequences spliced together through a series of complex processing events. Alternative splicing refers to the optional inclusion or exclusion of specific exons in transcripts derived from a **single gene** , which leads to structural and functional changes in the encoded proteins. Although many components of the machinery directing the physical excision of introns and joining of exons have been elucidated in recent years, the signaling pathways regulating the activity of the machinery remain largely unexplored. RESULTS: A calcium-mediated signaling pathway regulates alternative splicing at a specific site of human plasma membrane calcium pump-2 transcripts. This site consists of three exons, which are differentially used in a tissue-specific manner. In IMR32 neuroblastoma cells, a transient elevation of intracellular calcium changed the predominant pattern from one in which all three exons are included to the coexpression of a variant including only the third exon. Western-blot analysis demonstrated that the newly expressed mRNAs are faithfully translated. Once induced, the new splicing pattern was maintained over multiple cell divisions. Protein synthesis was not required to induce the alternative splice change, indicating that all components necessary for a rapid cellular response are present in the cells. CONCLUSIONS: Calcium signaling exerts a direct influence on the regulation of alternative splicing. Notably, a calcium-mediated change in the expression of **alternatively spliced variants** of a calcium regulatory protein was discovered. The change in splicing occurs quickly, is persistent but reversible and leads to a corresponding change in protein expression. The specific nature in which differently spliced protein variants are expressed, and now the fact that their expression can be regulated by distinct intracellular signaling pathways, suggests that the regulation of alternative splicing by physiological stimuli is a widespread regulatory mechanism by which a cell may coordinate its responses to environmental cues.

L23 ANSWER 13 OF 14 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 95327070 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7603452
TITLE: Alternatively spliced forms of the alpha subunit of the epithelial sodium channel: distinct sites for amiloride binding and channel pore.
AUTHOR: Li X J; Xu R H; Guggino W B; Snyder S H
CORPORATE SOURCE: Department of Neuroscience, Psychiatry, and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA.
SOURCE: Molecular pharmacology, (1995 Jun) 47 (6) 1133-40. Journal code: 0035623. ISSN: 0026-895X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-S78582
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950822
Last Updated on STN: 19950822
Entered Medline: 19950804

AB The amiloride-sensitive epithelial sodium channel (ENAC) consists of

New **alternatively spliced variants** of calmodulin-dependent protein kinase II from rabbit liver

AUTHOR: Takeuchi M; Fujisawa H (Reprint)
CORPORATE SOURCE: ASAHIKAWA MED COLL, DEPT BIOCHEM, ASAHIKAWA, HOKKAIDO 078851, JAPAN (Reprint); ASAHIKAWA MED COLL, DEPT BIOCHEM, ASAHIKAWA, HOKKAIDO 078851, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: GENE, (9 OCT 1998) Vol. 221, No. 1, pp. 107-115.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0378-1119.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 27

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Polymerase chain reaction analysis revealed four **alternatively spliced variants** of each of the gamma and delta isoforms of calmodulin-dependent protein kinase II (CaM-kinase II) in rabbit liver. Among the four Variants of the gamma isoform, two were novel ones, designated as CaM-kinase II gamma-H and gamma-I. The gamma-I variant possessed both of the two deletable exons, D2a and D2b, which had never been found together in any variant. Sequence analysis of the gamma-I indicated that the D2a was upstream of the D2b and that they were contiguous with each other in the gamma-I. Among the four variants of the delta isoform, two were also novel ones, designated as CaM-kinase II delta-11 and delta-12, and the other two were the already-reported ones, delta-2 and delta-6. The delta-11 and delta-12 were identical to the delta-2 and delta-6, respectively, except that three bases (CAG) located at a splicing junction was deleted in the delta-11 and delta-12, suggesting two splicing sites of a single intron. Thus, the diverse splicing patterns may produce many more variants than those so far

L16 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:80456 CAPLUS
DN 134:322202
ED Entered STN: 05 Feb 2001
TI Relationship between novel isoforms, functionally important domains, and subcellular distribution of CD164/endolyn
AU Chan, James Yi-Hsin; Lee-Prudhoe, Jane E.; Jorgensen, Britt; Ihrke, Gudrun; Doyonnas, Regis; Zannettino, Andrew C. W.; Buckle, Veronica J.; Ward, Christopher J.; Simmons, Paul J.; Watt, Suzanne M.
CS Medical Research Council Molecular Hematology Unit, Institute of Molecular Medicine, Oxford, OX3 9DS, UK
SO Journal of Biological Chemistry (2001), 276(3), 2139-2152
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
CC 6-3 (General Biochemistry)
Section cross-reference(s): 3, 13
AB Functional analyses have indicated that the human CD164 sialomucin may play a key role in hematopoiesis by facilitating the adhesion of human CD34+ cells to the stroma and by neg. regulating CD34+CD38lo/- cell proliferation. We have identified three novel human CD164 variants derived by alternative splicing of bona fide exons from a single genomic **transcription unit**. The predominant CD164(E1-6) isoform, encoded by six exons, is a type I transmembrane protein containing two extracellular mucin domains (I and II) interrupted by a cysteine-rich non-mucin domain. The 103B2/9E10 and 105A5 epitopes, which specify ligand binding characteristics, are located on the exon 1-encoded mucin domain I. Three human CD164(E1-6) mRNA species, exhibiting differential polyadenylation site usage, are differentially expressed in hematopoietic and non-hematopoietic tissues. This study provides addnl. evidence that human CD164(E1-6) represents the ortholog of murine MGC-24v and rat endolyn. Comparative anal. of murine MGC-24v/CD164(E1-6) with human CD164(E1-6) revealed two potential splice variants and a similar genomic structure. Whereas the human CD164 gene is located on chromosome 6q21, the mouse gene occurs in a syntenic region on chromosome 10B1-B2. By confocal microscopy, human CD164 in CD34+CD38+ hematopoietic progenitor (KG1B) and epithelial cell lines appears to be localized primarily in endosomes and lysosomes, with low concns. at the cell surface. However, in a minority of KG1B cells, CD164 is more prominently expressed at the plasma membrane and in the recycling endosomes, suggesting that its distribution is regulated in cells of hematopoietic origin.
ST CD164 isoform human gene cDNA sequence; mouse gene cDNA sequence CD164
IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(CD164; cloning, sequence and characterization of human and mouse **alternatively spliced variants** of protein CD164)
IT Cell adhesion
DNA sequences
Mouse (Mus musculus)
Protein sequences
RNA splicing
cDNA sequences
(cloning, sequence and characterization of human and mouse **alternatively spliced variants** of protein CD164)
IT Genetic element
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(exon; cloning, sequence and characterization of human and mouse **alternatively spliced variants** of protein

CD164)
IT Chromosome
(human 6, 6q21; chromosomal location of human CD164)
IT Cell membrane
Endosome
Hematopoietic precursor cell
Lysosome
(localization of human CD164 in hematopoietic progenitor cells)
IT Chromosome
(mouse 10, 10B1-B2; chromosomal location of murine CD164)
IT 217307-88-5, Sialomucin CD164 (human precursor) 252243-26-8
291801-42-8 336652-07-4, Protein CD164 (human isoform δ 4)
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(amino acid sequence; cloning, sequence and characterization of human and mouse **alternatively spliced variants** of protein CD164)
IT 317799-60-3, GenBank AF299340 317799-61-4, GenBank AF299341
317799-62-5, GenBank AF299342 317799-63-6, GenBank AF299343
317799-64-7, GenBank AF299344 317799-65-8, GenBank AF299345
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; cloning, sequence and characterization of human and mouse **alternatively spliced variants** of protein CD164)
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

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95359262 EMBASE

DOCUMENT NUMBER: 1995359262

TITLE: Characterization of RET proto-oncogene 3' splicing variants and polyadenylation sites: A novel C-terminus for RET.

AUTHOR: Myers S.M.; Eng C.; Ponder B.A.J.; Mulligan L.M.

CORPORATE SOURCE: Department Pathology and Paediatrics, Queen's University,Kingston, Ont. K7L 3N6, Canada

SOURCE: Oncogene, (1995) 11/10 (2039-2045).
ISSN: 0950-9232 CODEN: ONCNES

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 022 Human Genetics
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The RET proto-oncogene, which encodes a receptor tyrosine kinase, displays **multiple alternative splicing variants** . Splicing of sequences 3' of **exon** 19 to generate several coding and untranslated region (UTR) sequences has been previously reported. We have sequenced the full length RET coding region and characterized the transcripts and 3' UTRs generated by alternative splicing of the RET 3' terminus. These analyses were performed using both RET cDNA cloned from a pheochromocytoma library and reverse transcriptase PCR products generated using RNA from a neuroblastoma cell line (LA-N-2). Three different carboxyl termini were identified. In addition to the nine and 51 terminal amino acid forms already known, we identified a third with 43 terminal amino acids predicted to encode a novel RET protein isoform. A total of 3621 base pairs of DNA 3' of exon 19, which spans the alternatively spliced exons and RET UTRs, was sequenced. Four polyadenylation sites were identified. The observed combinations of polyadenylation sites and 3' coding sequence suggest that RET transcripts with up to 10 different 3' sequences and up to 40 different full length RET transcripts may exist.

PREV199900080655

TITLE: Identification of **multiple** alternative **splice variants** of the KOR-3/ORL-1 receptor gene that are expressed differentially in mouse brain.

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SOURCE: Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 1595. print.
Meeting Info.: 28th Annual Meeting of the Society for Neuroscience, Part 2. Los Angeles, California, USA. November 7-12, 1998.
ISSN: 0190-5295.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)

LANGUAGE: English

ENTRY DATE: Entered STN: 1 Mar 1999
Last Updated on STN: 1 Mar 1999

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L84: Entry 28 of 47 File: USPT Feb 10, 2004

DOCUMENT-IDENTIFIER: US 6691042 B2
TITLE: Methods for generating differential profiles by combining data obtained in separate measurements

Detailed Description Text (124):
In specific embodiments of the invention, when an exon has alternative spliced variants, a set of polynucleotide probes of successive overlapping sequences, i.e., tiled sequences, across the genomic region containing the longest variant of an exon can be included in the exon profiling arrays. The set of polynucleotide probes can comprise successive overlapping sequences at steps of a predetermined base intervals, e.g. at steps of 1, 5, or 10 base intervals, span, or are tiled across, the mRNA containing the longest variant. Such set of probes therefore can be used to scan the genomic region containing all variants of an exon to determine the expressed variant or variants of the exon to determine the expressed variant or variants of the exon. Alternatively or additionally, a set of polynucleotide probes comprising exon specific probes and/or variant junction probes can be included in the exon profiling array. As used herein, a variant junction probe refers to a probe specific to the junction region of the particular exon variant and the neighboring exon. In a preferred embodiment, the probe set contains variant junction probes specifically hybridizable to each of all different splice junction sequences of the exon. In another preferred embodiment, the probe set contains exon specific probes specifically hybridizable to the common sequences in all different variants of the exon, and/or variant junction probes specifically hybridizable to the different splice junction sequences of the exon.

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26. (original) The method of claim 19, wherein multiple concentrations of the compound are tested at multiple time points.

27. (new) A method of determining a cellular or tissue response to a compound or external stimulus comprising:

a) contacting a compound or external stimulus with a cell or tissue for a sufficient time to allow transcription of a gene to generate an RNA,

b) isolating total RNA from the cells or tissue after sufficient time to allow transcription to occur;

c) hybridizing the isolated RNA preparation to said plurality of different nucleic acids of the solid substrate of claim 3 under conditions wherein an individual RNA transcript of the ~~isolated RNA preparation~~ hybridizes to a specific nucleic acid;

d) determining the change in level of expression of a set of IRCs encoded by said RNA containing at least 50% of the IRCs ~~as set forth in Table 1~~;

e) repeating steps a) through d) at least three times; and

f) determining which IRCs show a statistically significant level of change based on the at least three replicate steps of step e), wherein the IRCs so identified constitute an IRC panel.

28. (new) The method of claim 27, wherein the determining is performed by a method selected from the group consisting of a microarray, a dot blot hybridization, a slot blot hybridization, a Real-time PCR using SYBR Green detection, a Real-time PCR using TaqMan assay, a Real-time PCR using hybridization probes, a Real-time PCR using molecular beacons, a quantitative competitive PCR, a northern blot analysis, a RNase nuclease protection assay and a S1 nuclease protection assay.

29. (new) A method of determining the toxicity of a compound comprising:

a) contacting a cell or target tissue with a candidate compound;

b) isolating total RNA from the cell or target tissue after sufficient time to allow transcription to occur;

c) hybridizing the isolated RNA preparation to said plurality of different nucleic acids of the solid substrate of claim 3 under conditions wherein an individual RNA transcript of the isolated RNA preparation hybridizes to a specific nucleic acid;

d) determining the change in level of IRCs as set forth in the panel of IRCs of claim 27; and

e) comparing the readout with the effect of known toxicological agents on said panel of IRCs for that cell or target tissue.

30. (new) The method of claim 29, wherein the determining is performed by a method selected from the group consisting of a microarray, a dot blot hybridization, a slot blot hybridization, a Real-time PCR using SYBR Green detection, a Real-time PCR using TaqMan assay, a Real-time PCR using hybridization probes, a Real-time PCR using molecular beacons, a quantitative competitive PCR, a northern blot analysis, a RNase nuclease protection assay and a S1 nuclease protection assay.

31. (new) A method of determining the global profile of physiological changes or the signaling responses of cells (global IRC) treated with a candidate compound or exposed to an external stimulus compared to untreated or unstimulated cells, comprising:

a) contacting a batch of untreated or unstimulated test cells with either a candidate compound or exposing the cells to an external stimulus;

b) isolating total RNA from the cells after sufficient time to allow transcription to occur;

c) hybridizing the isolated RNA preparation to said plurality of different nucleic acids of the solid substrate of claim 3 under conditions wherein an individual RNA transcript of the isolated RNA preparation hybridizes to a specific nucleic acid;

d) determining the change in level of expression of a set of IRCs selected from Table 1 or as identified by the method of claim 27, encoded by said RNA in both the treated and untreated cells; and

e) comparing the level of expression of said set of IRCs from both the treated and untreated cells to an IRC panel identified by the method of claim 27; and
wherein the global IRC profile is determined by the RNA transcripts that exhibit a change

in expression between treated and untreated cells.

32. (new) A method of determining the global profile of physiological changes or the signaling responses of cells (global IRC) treated with a candidate compound compared to a standard compound having known effects on the cell system under study comprising:

a) contacting a separate batch of test cells with either a candidate compound or a standard compound for which a global IRC profile has been determined;

b) isolating total RNA from the cells after sufficient time to allow transcription to occur;

c) hybridizing the isolated RNA preparation to said plurality of different nucleic acids of the solid substrate of claim 3 under conditions wherein an individual RNA transcript of the isolated RNA preparation hybridizes to a specific nucleic acid;

d) determining the changes in the level of expression of IRCs encoded by said RNA of a predetermined set of IRCs identified by the method of claim 27; and

e) comparing the global IRC profile obtained with the test compound to the global IRC profile obtained with the standard compound.

33. (new) The method of claim 32, wherein the IRCs used to determine the global IRC profile are selected from Table 1.

34. (new) The method of either of claims 31 or 32, wherein the determining is performed by a method selected from the group consisting of a microarray, a dot blot hybridization, a slot blot hybridization, a Real-time PCR using SYBR Green detection, a Real-time PCR using TaqMan assay, a Real-time PCR using hybridization probes, a Real-time PCR using molecular beacons, a quantitative competitive PCR, a northern blot analysis, a RNase nuclease protection assay and a S1 nuclease protection assay.

35. (new) The method of any one of claims 27, 29, 31, or 32, wherein the determining step is performed by hybridizing the isolated RNA preparation with a set of 50 to 500 different nucleic acids, the set of nucleic acids are coupled to a solid substrate in known locations, each different nucleic acid of the set of nucleic acids

hybridizes with at least a portion of an RNA transcript, and wherein the set of nucleic acids hybridize with at least 80% of the intrinsic reporters of cell signaling listed in Table 1, or orthologs thereof.

36. (new) The method of claim 35, wherein each different nucleic acid is contained at least in triplicate on the solid substrate in a different known location.

37. (new) The method of claim 36, wherein the genes that are identified have an increased relative level of expression in the presence of the agent that is 1.5 fold or greater.

38. (new) The method of claim 37, wherein the contacting time is 1 hour at 37°C.

39. (new) The method of claim 37, wherein the determining step includes the use of a data analysis algorithm based on thresholding by fold-change and t-test value determination.

40. (new) The method of claim 39, further comprising the steps of:

performing Real-term PCR for the IRCs identified in step (d); and

determining the amount of PCR product wherein an IRC is identified to have an increased relative level of expression in the presence of the compound or external stimulus relative to in its absence when the amount of PCR product determined increases.

41. (new) The method of claim 40, further comprising:

contacting multiple concentrations of the compound or external stimulus with the cells at multiple times of incubation;

determining the individual rate of transcription and/or the amount of transcription of the IRCs; and

identifying the IRCs within the panel of IRCs that show a difference in the level of expression in the presence of the compound or external stimulus relative

to in the absence of the compound or external stimulus at each time and each concentration; wherein the IRCs are identified that have a different level of expression in the presence of the compound or external stimulus at multiple times and multiple concentrations.

L78: Entry 8 of 13 File: PGPB Sep 12, 2002

DOCUMENT-IDENTIFIER: US 20020127615 A1
TITLE: TRAF-3 deletion isoforms and uses thereof

Brief Description of Drawings Paragraph:
[0026] FIG. 19. RNAse protection analysis of TRAF-3 mRNA splice variant expression. (A) A schematic representation of the TRAF-3 genomic sequence, with exons represented as wide black bars, is mapped onto the protein domains encoded by full-length TRAP-3 cDNA. Below are depicted the protected probe fragments resulting from hybridization of an anti-sense probe corresponding to the full-length TRAF-3 mRNA with all previously identified alternative splice forms of TRAF-3 mRNA. As indicated, most "secondary" protected fragments, may have contributions from multiple splice-variants. (B), RNAse protection of the indicated samples was performed using an antisense human GAPDH probe as described in Materials and Methods. The indicated band (absent from the yeast mRNA sample) corresponds to the expected 316 nt protected fragment resulting from hybridization of the probe with GAPDH mRNA. (C), TRAP-3 splice variant RNAse protection assay for Yeast tRNA (negative control), Ramos CC total RNA (negative control), and Jurkat D1.1 total RNA (positive control) using indicated probes. (Full-length TRAF-3 probe is indicated by "FL".) (D), TRAF-3 splice variant RNAse protection assay for BJAB, Daudi, and Raji total RNA using the indicated probes. In (C) and (D), diluted probes were run as size markers at the left and right of each gel. Displayed below each gel is the signal from each sample resulting from hybridization of probes to the internal control sense TRAF-3 RNA. Primary fragments corresponding to hybridization of a given probe with its complementary mRNA splice-variant are indicated by an asterisk (*). The asterisk in each .DELTA.130 lane marks the expected position for the .DELTA.130 primary fragment. An unexpected TRAF-3 specific band with an approximate size of 165 nt was protected by the probes FL, .DELTA.25, .DELTA.27, .DELTA.52, .DELTA.56, and .DELTA.83 and is indicated by a cross (). Data shown are representative of three independent experiments.

Detail Description Paragraph:
[0174] Isolated PAC clones were sequenced manually by cycle sequencing (dsDNA Cycle Sequencing System.RTM., Life Technologies). Sequence reactions were analyzed by denaturing PAGE (6% polyacrylamide/7 M Urea) and autoradiography (BioMax MS.RTM. film, Eastman Kodak). DNA fragments, cloned into the pCR2.0 or pCR2.1 TA cloning vector (Invitrogen) or the pBluescript SK(+) cloning vector (Stratagene), were sequenced on a automated ABI 373 DNA sequencer, by the DNA sequencing core facility at Columbia University or at the Molecular Resource Facility, New Jersey Medical School, UMDNJ. All sequence information was submitted to the BLAST server available through the National Center for Biotechnology Information (NCBI) (http://www.ncbi.nlm.nih.govlBLAST) or the DNA analysis programs of the Genetics Computer Group (GCG, Madison, Wis.) for alignment with additional sequences, identification of repetitive elements or any other homology. Unique sequences were selected and submitted to the Primer3 server available through the Whitehead Institute for Biomedical Research/M.I.T. Center for Genome Research (http://www-genome.wi.mit.edu/cgi-bin/primer/primer3.cgi) or Amplify 1.2 (University of Wisconsin, Madison, Wis.) to design oligonucleotides (obtained from Life Technologies) used for PCR or sequencing.

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